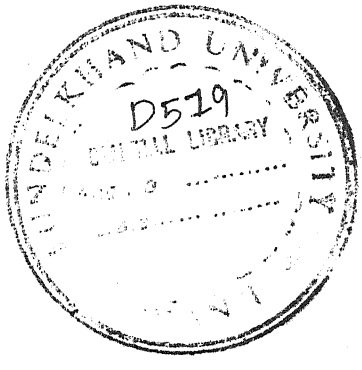
CLINICAL FEATURES AND RISK FACTORS IN PATIENTS OF STROKE WITH SPECIAL REFERENCE TO HYPERTENSION, ITS MANIPULATION AND OUTCOME

THESIS

FOR

DOCTOR OF MEDICINE
(INTERNAL MEDICINE)



BUNDELKHAND UNIVERSITY
JHANSI (U.P.)

2005 **SAURABH TANDON**

Dedicated

To

My Parents

CERTIFICATE

This is to certify that the work entitled ***"Clinical features and risk factors in patients of stroke with special reference to hypertension, its manipulation and outcome"*** which is being submitted as a thesis for M.D. (Internal Medicine) Examination 2005 of Bundelkhand University, Jhansi, has been carried out by ***Dr. Saurabh Tandon*** in the Department of Medicine, M.L.B. Medical College, Jhansi.

This method described was undertaken by the candidate himself and the observations recorded have been periodically checked up. He has put in the necessary stay in the Department as per University regulations, and has fulfilled the conditions required for the submission of thesis according to University regulations.

Dated: 9/11/04

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Professor & Head,
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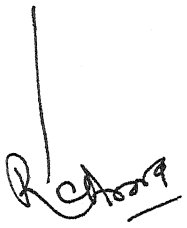

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Acknowledgement

Today as I write this acknowledgement I do so with a deep sense of gratitude to my esteemed teacher and ***Guide Professor R.C. Arora****,* ***M.D.,D.Sc****, Ex. Professor and Head, Department of Medicine, M.L.B., Medical College, Jhansi for having guided me throughout this thesis work. A brilliant teacher and an eminent academician, it has been my proud privilege to have been associated with him and to work under his guidance. I express my reverent gratitude to him for his able guidance, sound supervision and valuable suggestions without which it would have not been possible to complete this work.*

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for his valuable suggestions and keen observance, encouragement and help which made this work presentable.

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I express my deep sense of gratitude to my learned and honourable teachers ***Dr. Praveen Jain M.D. D.M****, and* ***Dr. N.S. Sengar M.D. D.M.****, Department of Medicine for giving me inspiration and encouragement throughout my residency period.*

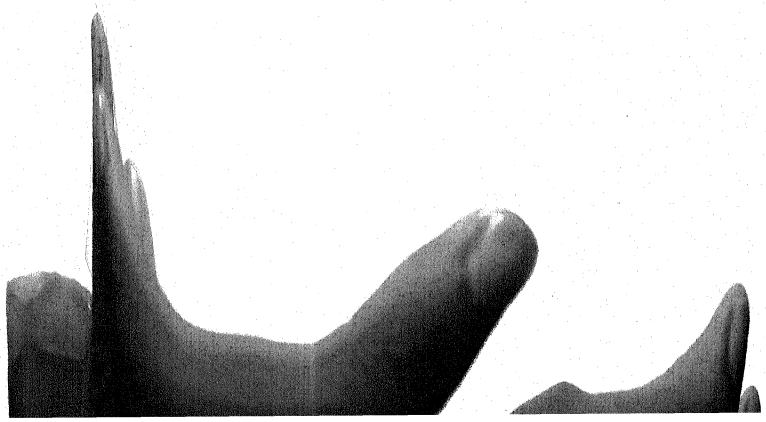
I am indebted to my Parents for their love and encouragement, which was bestowed upon me by their good wishes during the study.

Lastly I would like to thank all those unnamed subjects who were a part of this study.

Saurabh Tandon

Date 9/11/04 ***Saurabh Tandon***

Contents



CONTENTS

Introduction

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Stroke is defined as a sudden onset of non convulsive focal neurological deficit (or global) and lasting for more than 24 hrs[1].

Stroke is the third leading cause of death worldwide and accounts for 10-20% of deaths in industrialized countries[2]. Worldwide, about 20 million people suffer from stroke each year; 5 million die as a consequence and 15 million will survive, of those, who survive, 5 million will be disabled by their stroke.[3] Therefore, the global burden of stroke needs to be defined for the developed and the developing nations. For example, it is estimated that approximately 4.5 million Americans are currently living with the effects of stroke, and that every year another 570,000 will survive a stroke that will result in disability.

Global Burden of Diseases (GBD) Study,[4] in 1990, reported 9.4 million deaths in India of which 61,900 were from stroke and the disability adjusted life years (DALYs) lost almost amounted to 28.5 million- nearly six times higher than that due to malaria. When these estimates were projected for the year 2020, Murrey and Lopez reported that 61 million DALYs are likely to be lost due to stroke, of these 52 million (84%) will be in the developing countries.[5]

Reddy and Yusuf have reemphasized the "Health Care and Economic Consequences" of emerging epidemic of cardiovascular diseases in developing countries.[6]

India will face an enormous socio-economic burden to meet the costs of rehabilitation of "stroke victims" because the population is now surviving through the peak years (age 55-65) of occurrence of stroke (CVD).

However, for stroke prevention planning, reliable epidemiological information on pattern of disease and exposure to major risk factors and morbidity and mortality trends for CVD in defined populations is not available. Recent community surveys for "hemiplegia" presumed to be CVD, identified 320 cases in 145,456 persons, indicating an overall Crude Prevalence Rate (CPR) of 220 per 100,000 persons[7] another recent survey on 20,842 rural residents in East India report a CPR for stroke in elderly (age 41-60 yrs) at 540/100,000.[8]

Furthermore in two prospective stroke studies, during the period 1963-1968 and 1978-1982 in Mumbai, using identical methodologies, it was observed that their was a significant drop in case fatality rate (32% to 12%) thereby resulting in a higher survival (68% to 88%) but with residual disability. Thus, these changing trends have posed a major social challenge in occupational

rehabilitation and in solving the needs for stroke survivors.[9] these data suggest that India is already facing "Stroke Epidemic."

In addition, published reports suggest that CVD occurs at all ages in both sexes and with increasing frequency with advancing age. Prospective studies on acute stroke have shown that hypertension, diabetes mellitus, low normal hemoglobin and tobacco use (smoking/chewing) are important risk factors.[10-13]

Hastak, et al (2003) reported that at 28 days the overall case fatality rate was 9% and nearly 31% of survivors had severe neurologic disability/handicap whereas 13% had mild disability needing assistance. Only 47% of survivors were independent at the end of 28 days; here 17% were not aware of having hypertension.[14] though such information being selective does not represent stroke morbidity/mortality patterns but it does show the current trends in DALYs in stroke population in the respective communities.

Thus to design stroke prevention strategies, public awareness and health education on warning symptoms of hypertension and Transient Ischemic Attacks (TIAs) by media is optimal. Lifestyle changes, dietary habits and intensive campaign against tobacco use will prove rewarding. Primary health care teams should receive training on nomenclature and in bedside diagnosis, in the absence of CT facilities in rural and remote areas. Mass screening surveys to

identify "hypertensives" and "stroke prone" subjects, wherever feasible, should be undertaken.

Overall 85-90% are ischaemic and 10-15% are haemorrhagic.[15,16] In India so far two studies have been done regarding stroke, revealing an incidence of 13-33 per 100,000.[17] The highest Crude Prevalence Rate of hemplegia presumed to be due to stroke has been seen in Mumbai (Parsi community) 943 per 100,000.[18]

Various modifiable and non-modifiable risk factors like age, race, gender, genetic factors and hypertension,dyslipidemia, diabetes, ischaemic and valvular heart disease predispose to stroke.

Physiological derangement, such as high blood pressure, hyperglycemia, pyrexia and intracranial hypertension, is common in acute stroke and associated with poor outcome.[19] There is now a worldwide interest in trying to reduce poor outcomes after stroke through effective management of these conditions.

Hypertension is a major risk factor in all races, both sexes and all age groups for stroke. The risk of stroke in a patient with diastolic blood pressure>110 mm of Hg is fifteen fold than that of a patient with diastolic pressure of <80 mrn of Hg.[20] Other risk factors that could indirectly influence the prevalence of stroke in a population are

lack of awareness of primary hypertension and access to health care. Asians have higher incidence of stroke as compared to whites.[21]

The disease most commonly occurs in middle and later years of life with males making up about 61% of the stroke cases and most cases occurred in winters about 63% of the total. Majority of the patients get admitted with a definite history of focal neurological deficit (76.1%), comatosed (32.6%), and 16.2% had a previous history of Trans Ischaemic Attack. In a study, amongst the risk factors hypertension was the commonest (71.3%), ischaemic heart disease (12.6), diabetes mellitus (9.2%), valvular heart disease (4.3%) and pregnancy (1.7%).[22]

Acute stroke, whether due to infarction or hemmorhage is associated with hypertension in 75% of patients, of whom 50% have a previous history of high BP. Early hypertension is associated with a poor outcome,[23-25] particularly in patients with impaired consciousness.[26] Evidence is now accumulating that low systolic blood pressure is also associated with a poor outcome; as a result, a 'U-shaped' curve appears to relate outcome and BP with the best outcome observed in normotensive and mildly hypertensive patients.

Manipulation of blood pressure in stroke may improve outcome.[27] Despite various studies, data on the prognostic significance of early BP in stroke remains unclear. BP values may

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not reliably reflect any impact of BP on stroke outcome. They also suggest a potential differential effect of BP manipulation: increasing or decreasing BP may be beneficial for patients with BP extremes in one direction, but detrimental for those with BP values in the opposite direction.[27]

A spontaneous increase in BP is common in the setting of acute ischemic stroke in humans.[28-31] the optimal management of elevated BP pressure, which may be labile, is still a common clinical concern in a patient presenting with cerebral infarction. Neurological deterioration has been frequently associated with the use of antihypertensive agents.[32-39]

Despite early reports of improved outcome in patients with ischemic stroke treated with induced hypertension,[40-42] this practice has been confined to a handful of stroke units, and results have been rarely reported. Induced hypertension in acute stroke may be associated with adverse effects such as hemorrhage into infarct, cerebral edema, and myocardial ischemia in the patient with concomitant artherosclerotic coronary artery disease. Despite these concerns, induced hypertension is now recommended to improve cerebral perfusion in the treatment of cerebral ischemia due to vasospasm after SAH and in maintaining cerebral perfusion pressure in patients with posttraumatic brain edema.

After SAH, hypertension can produce clinical improvement with minimal and acceptable systemic toxicity.[43] In such patients induced arterial hypertension increases Cerebral Blood Flow (CBF)[44,45] and can improve vasospasm related ischemic neurological deficits. Recent data from PET[46] and MRI[47] studies in patients with ischemic stroke combined with clinical observations suggest that a particular situation may exist in some acute stroke patients.

Blood pressure is frequently elevated early after acute stroke, an observation most commonly thought to be explained by mental stress, previous hypertension, increased catecholamine secretions or a multitude of several factors[48-51] Guidelines for the management of acute stroke suggest this increase in BP should not be treated, as it generally declines after a few days. Pharmacological intervention is only recommended in patients with extremely high BP values, and caution is required to avoid excessive BP drop. [52-54] One concern with lowering BP in the acute phase of ischemic stroke is that it may further compromise any critical perfusion in the area of tissue at risk for cerebral infarct.

Cerebral perfusion pressure, a function of systemic BP and intracranial pressure, is one of the factors determining the final infarct volume.8 Conversely, very high BP may enhance infarct related edema or induce hemorrhagic conversions of an infarct.

Review Of Literature

Review of literature

Parashar BS, Kaushik NK[55] in their study of Stroke in High Altitude concluded that out of 100 cases average age was 57.88 yrs, maximum incidence in 7th decade, with M: F 1.95:1, 96% were anterior circulation stroke, with 69% infarct and 26% had PICH. Risk factors could be identified in 91% cases. Hypertension was present in 62%, smoking in 60% and both hypertension and smoking were present in 36%, diabetes mellitus in 9%, 10% had h/o previous stroke. 83% presented with hemiparesis, 7th nerve palsy in 74%, speech disorders in 46% and altered sensorium in another 28%.

Deobarh G. Stewart, MD[56] death rates from stroke have been declining with an annual decrease of 5% per year in the United States since 1970.2 the incidence of first stoke rises exponentially with age. In the 55 to 59 year old age group, the risk of stroke is about 5% per year, whereas in the 80 to 84 year old group the risk is about 25% per year.[21] gender has been shown to affect stroke risk; men have a 30% increased risk until the later decades of life when women have a higher risk. [21] More women under the age of 45 years die from stroke than myocardial infarction.

A disproportionate percentage of subarachnoid hemorrhage occurs in women. Race related differences in incidence and mortality of stroke have also been demonstrated. When compared with whites, blacks have a higher incidence and mortality of stroke, which may be in part attributable to a relatively late presentation for medical care.[21,57]

Helen M Dewey Jonathan Sturm et al,[58] made a study to determine the incidence and outcome of sub-types of cerebral infarction (CI). Annual incidence rates per 100,000 persons adjusted to the world population were 11 Total Anterior Cerebral Infarction (TACI), 25 for PACI, 17 for POCI and 18 for LACI. 28-day case fatality was highest for TACI 35%, and first year recurrence rate was highest for PACI 17%; TACI had the poorest outcome at 3 and 12 months.

Rahman K M et al[22] conducted a study in 85 consecutive stroke patients irrespective of age and sex admitted during the period of August 2000 to June 2001. The outcome was that about of the admitted cases 81.8% were males and were of the age group 62.54 +/- 13.08 yrs. and 18.82% were females of the age group 58.81+1-12.27 yrs. 78.82% were hypertensive and had hemiplegia on presentation. Altered consciousness was more common when hemorrhagic stroke 54.84%.

G.Q.Khan et al[59] studying stroke admissions in Kashmir Valley concluded that intracerebral haemorrhage shared a greater percentage. The amazing fact was that 635 (23.9%) who were clinically suspected of having ischaemic strokes were documented as small intracerebral haemorrhage on CT Scan of head. Again, most of the patients who were admitted in a comatosed state and died immediately were also clinically suspected as having intracerebral haemorrhage. Hypertension, rupture of AV malformations, and intraparenchymal aneurysms are the leading factors responsible for intracerebral haemorrhage. Among them hypertension is the most common, the most modifiable and the most important risk factor which needs multipronged approach.

Daad H. Akbar, Maunona Mushtaq[66] in their study of clinical profile and risk factors in patients of stroke in Saudi Arabia out of 103 patients which form part of their study 34 (33%) had Dyslipidemia and this was more common in ischemic stroke (74%) as compared to non ischemic stroke.

James F. Meschia, M.D. and Thomas G. Brott, M.D[60] It now appears likely that hyperlipidemia is an independent risk factor for ischemic stroke.[61] Lipids tend to temporarily fall after an acute stroke.[62,63] This phenomenon is probably not strictly related to inadequate nutrition that might result from conservative early

management of dysphagia because the same phenomenon has been seen in patients with many acute non-neurologic conditions. Aull and colleagues have recommended that a fasting lipid profile should be obtained on the first or second morning after stroke to avoid pseudonormalized values.[62] Mendez and colleagues found in patients ages 60 to 69 that mean fasting LDL cholesterol levels changed from 136 ± 20 mg/dl on day 1 to 115 ± 17 mg/dl on day 7 and 160 ± 16 mg/dl at 3 months following an acute ischemic stroke.[63] The investigators concluded that lipids should be checked at least three months after a stroke to avoid missing hyperlipidemia in some patients.

Chirstensen H et al,[64] made a study to evaluate how soon after stroke the diagnosis of hypertension could be established. In a prospective study of 1192 patients with acute stroke within 6 hrs, blood pressure was measured serially at 2 hrs intervals during the first 24 hrs. In 779 patients with mild to moderate ischemic stroke or TIA, Mean Arterial Pressure (MAP) was 118 mm of Hg on admission and 109 mm of Hg 4 hrs later. No such early decrease was seen in 228 patients with severe cerebral infarction. BP 24 hrs after admission in patients with mild to moderate cerebral infarction or TIA was representative of patient's BP 3 months after stroke. A diagnosis of arterial hypertension could be established a few days after stroke.

The worldwide literature documents that intracerebral haemorrhage was responsible for 10-15% of all strokes and its incidence ranges from 10-20 per 100,000 populations.

Carlberg et al,[26] demonstrated that previous hypertension was the strongest predictor of elevated blood pressure in stroke patients on admission.

A 20 year follow-up of the National Health and Nutrition Survey conducted by **Qureshi A I et al**,[65] to evaluate long-term risk of stroke, type of stroke and predictors of stroke associated with Isolated Systolic Hypertension (ISH) and Borderline Isolated Systolic Hypertension (BISH) and this risk compares with that for persons with diastolic hypertension and normotension.

ISH defined as SBP >160 mm of Hg and DBP < 90 mm of Hg
BISH defined as SBP 140-159 mm of Hg and DBP < 90 mm of Hg
Relative Risk (RR) in ISH was 2.7 and RR in BISH was 1.4

Thus he concluded that increased risk of ischemic and intracerebral hemorrhage was observed in patients with BISH, similar to those with ISH and Diastolic hypertension (10).

Gasowski J et al[66] conducted a trial to explore the independent roles of pulse and mean pressure as predictors of mortality in a wide range of patients with hypertension. He concluded

that in hypertensive patient's high pulse pressure is associated with an increased risk of fatal events.

Carlberg B, Asplund K, Hagg E[26] patients with acute stroke are often found to have high blood pressures on admission. A study conducted to study the prognostic value of high blood pressure in acute stroke. The study included 85 patients with intracerebral hemorrhage and 831 patients with ischemic disease.

Results showed that high BP in alert stroke was not associated to increased mortality. Stroke patients with impaired consciousness showed higher mortality rates with increasing BP (12).

Boreas AM et al[27] studied the prognostic values of BP in acute stroke, BP were recorded by reviewing BP records of 817 patients who were admitted as cases of stroke using the mean of daytime as well as night time systolic and diastolic BP values. Only night time systolic BP 165 mm of Hg, night time diastolic BP 60 mm of Hg and a decrease in daytime diastolic BID between 0-4 days of 10 mm of Hg showed a significant relationship with poor outcome.

Control of hypertension in the acute phase of stroke has been studied in small trials and case reports.[64] Hypertension occurs in 75% of patients with infarction or hemorrhage. Two-thirds of these patients had preexisting hypertension. Current published recommendations are to gradually lower the blood pressure to

185/105 mmHg if the patient has a premorbid history of hypertension, and to 160/95 mmHg in patients with no history of hypertension. Patients with hemorrhagic stroke should probably be managed similarly, but the range of acceptable systolic and diastolic pressure can be 5 mmHg higher in each category mentioned above. Medications used for acute treatment of hypertension can include labetalol, enalapril and clonidine.

Danilo Toni, Marco Fiorelli et al,[68] made a study to identify predictors of early neurological improvement in acute ischemic stroke patients, to evaluate its impact on clinical outcome. Thirty-four patients (22%) improved, 84 (56%) remained stable, and 34 (22%) deteriorated.

In the **International Stroke Trial**, involving 17,398 patients with ischemic stroke, 80% of patients had high blood pressure; the mean systolic blood pressure across the study was 160 mmHg.[69]. The natural history is for blood pressure to start falling within hours of stroke onset[70] and to settle over the first weeks after stroke[49, 51,71] few patients have a low blood pressure, as defined by systolic blood pressure <100 mmHg. Indeed, in the International Stroke Trial, less than 5% of patients with ischemic stroke had a systolic blood pressure < 120 mmHg.

Several case studies have reported on the results of actively lowering blood pressure in acute stroke. The largest study in ischemic stroke (n=481) used a variety of drugs so class-specific interpretation is not possible; [72] nevertheless, fall in blood pressure of 20-30% by day 2 were associated with improved outcome and reduced cerebral oedema. Similarly, the largest series in PICH (n=167) reported that lowering blood pressure was beneficial.[73] Other and far smaller case series have found that lowering blood pressure may be detrimental.[74-76]

Guy Rordorf, Steven C, Cramer[77] in their study to determine whether induced hypertension in stroke is safe and examined its effect on neurological deficits in patients presenting with cerebral infarction. They retrospectively reviewed all patients with the diagnosis of ischemic stroke over a period of 2.5 years. Thirty-three patients were not given a pressor, while 30 were treated with vasopressor in an attempt to improve cerebral perfusion.

Niaz Ahmed, Nils Gunnar Wahlgren[78] Intravenous Nimodipine West European stroke Trial (INWEST) enrolled acute ischemic stroke patients within 24 h (n=295) to the following groups: placebo (n=100), 1mg/h nimodipine (n=101) or 2mg/h nimodipine (n=94). Results were: no significant difference in BP was observed between the Total Anterior Circulation Infarct (TACI) and non-TACI

subtypes at baseline, nor did the subtypes differ in BP course within the treatment groups. For TACI patients, there was no outcome difference between the placebo and nimodipine treated groups. In multivariate analysis for TACI patients, BP reduction and nimodipine treatment had no relation with outcome. Systolic BP reduction was associated with a better clinical outcome.

They concluded that BP lowering and nimodipine treatment had no significant effect on outcome for TACI patients.

Aims

&

Objectives

Aims and objectives

Study the clinical profile and risk factors in patients of stroke admitted through emergency and OPD in the department of medicine of MLB Medical College, Jhansi. Study the effect of lowering and elevation of blood pressure on the clinical outcome of these patients.

Material & Methods

Material and methods

The present study was done on subjects presenting to medicine OPD and emergencies and admitted to medicine wards of Maharani Laxmi Bai medical College, Jhansi during the period from August 2003 to September 2004.

The study has been done on patients admitted through OPD and emergency in Maharani Laxmi Bai Medical College Jhansi, hospital presenting with signs of focal neurological deficit lasting for more than 24 hrs.

Criteria for selection

Any individual presenting with sudden/acute onset of focal neurological deficit indicating towards a vascular cause will be included and will be evaluated further on the following lines.

1. Detailed history and clinical examination
2. Determining the following risk factors by investigations.

A. Hypertension

i. Blood pressure measurement on admission and 2hrly thereafter for the first 24 hrs to estimate the mean systolic

and diastolic blood pressure. They would be then categorized as under:

Classification of Blood Pressure

Category	SBPmmHg		DBPmmHg
Normal	<120	and	<80
Pre hypertension	120-139	or	80-89
Hypertension, Stage1	140-159	or	90-99
Hypertension, Stage 11	>or--160	or	>or=100

B. Diabetes Mellitus

Random Capillary Blood sugar (RCBS) & fasting blood sugar and 2hrs PP blood sugar estimation would be done. The patient would be diagnosed as a case of Diabetes Mellitus if any of the three conditions were met:

Symptoms of diabetes i.e. polyuria, polydypsia, polyphagia, plus

RCBS >200mg/dl.

Or

Fasting plasma glucose> 126mg/dl

Or

Two hours plasma glucose>200mg/di during a GTT

B. Ischemic heart disease

ECG: To look for any evidence of myocardial infarction or ischemic changes.

Computed Tomographic scan of Head:

i) To know the cause of stroke whether hemorrhagic or infarct

ii) To know the site and extension of hemorrhage or infarct.

iii) To look for any evidence of previous infarct or hemorrhage.

C. Dyslipidemia

Fasting blood samples of patients would be evaluated for lipid profile status

Manipulation of Blood pressure:

The second part of my work involves manipulation of blood pressure in the form of elevation of blood pressure and lowering of blood pressure.

Selection criteria of patients for the study group and control group for pharmacological elevation of blood pressure:

Patient's (Group1) who develop fall in blood pressure and neurological deterioration within 48 hrs of admission as evident on clinical examination (having Mathew score of <65).[103]

The MABP of these patients would be determined from the formula:

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The MABP of these patients would be determined from the formula: (SBP+2DBP)/3

The patients would be divided into two groups i.e. study and control group1. The blood pressure would be elevated in the study group. This would be done using i.v. fluids, vasopressor, {phenylephrine in the dose (20-300 microgm/min)} over a period of 5 days.

The two groups would be then compared using unpaired 't" test and the Degree of freedom and the 'p' value would be calculated from the table. Thus, the significance of blood pressure elevation and the outcome would be interpreted as significant or not.

The second part of manipulation involves lowering of blood pressure. The study group would comprise of patients having blood pressure $\geq$ 220/110 mmHg even after 48 hours of admission. All patients in this group would undergo lowering of blood pressure by nimodipine and other antihypertensive drugs used orally.

The clinical outcome would be studied after 5 days of lowering of blood pressure. The patients blood pressure before and after lowering would be studied using paired 't' test and t value and DF would be determined. Thus the significance of clinical effect of lowering of blood pressure on the outcome of the patients would be studied.

Observations

Table 1

Distribution of cases according to sex

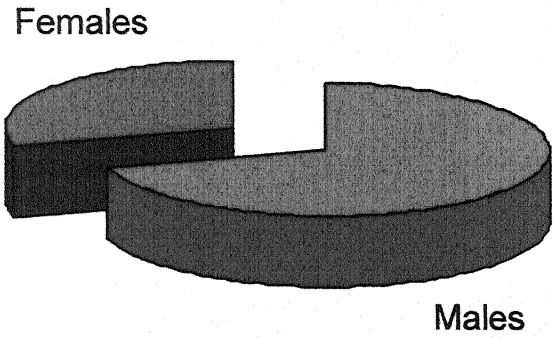
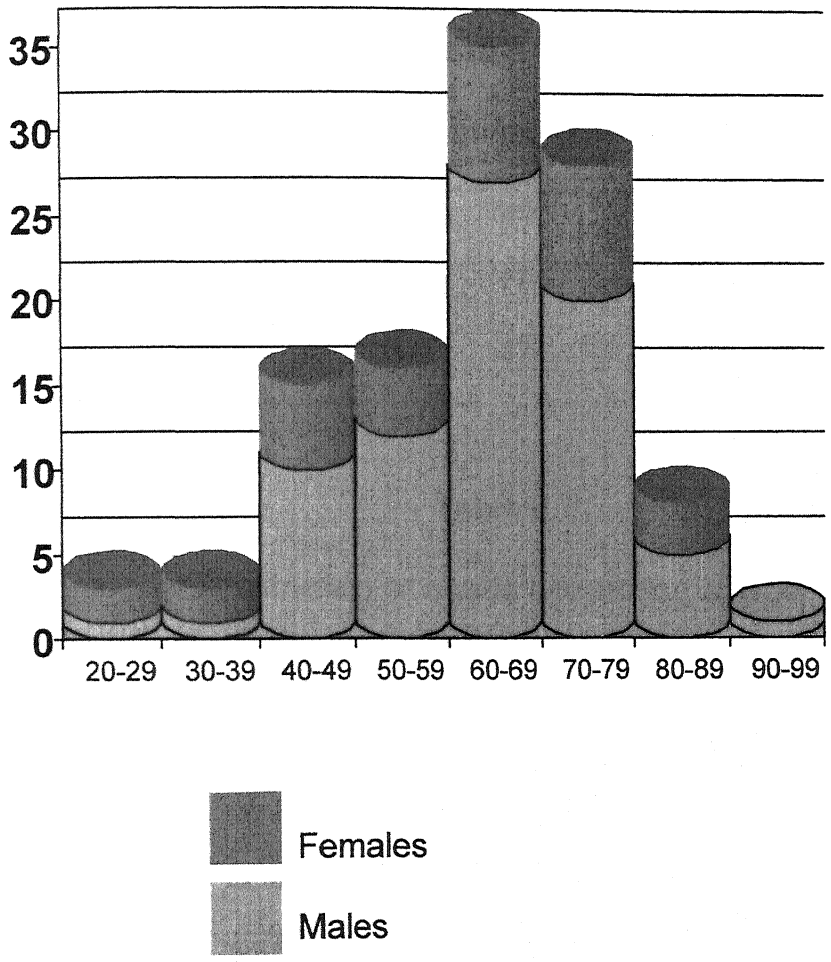


Table 2

Distribution of cases according to age group



Observations

The study comprised of n=109 patients. The various observations made are as follows:

Table I

Distribution of patients according to sex

	(n)	Percentage
Male	77	70.6
Females	32	29.35

Table II

Distribution of cases according to age

Age in years	No. of patients
20-29	3
30-39	3
40-49	15
50-59	16
60-69	35
70-79	28
80-89	8
90-99	1

Table 3

Clinical features at presentation

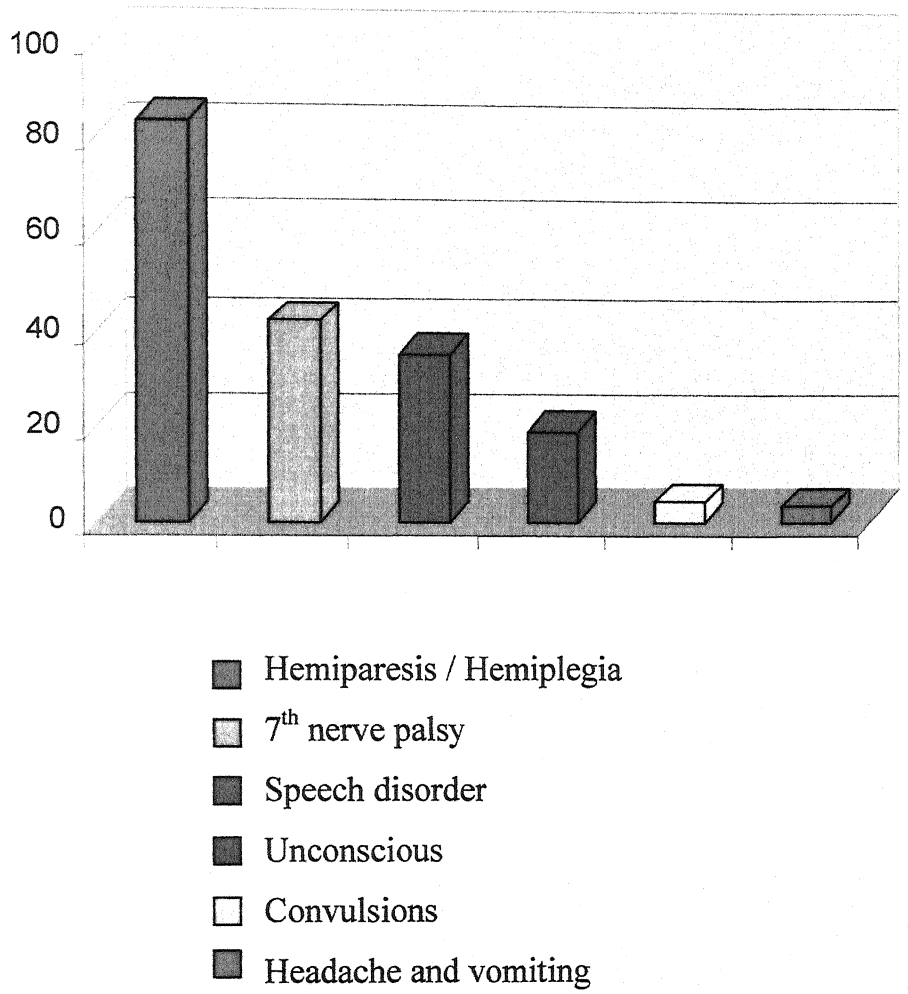


Table 4

Distribution of stroke subtypes

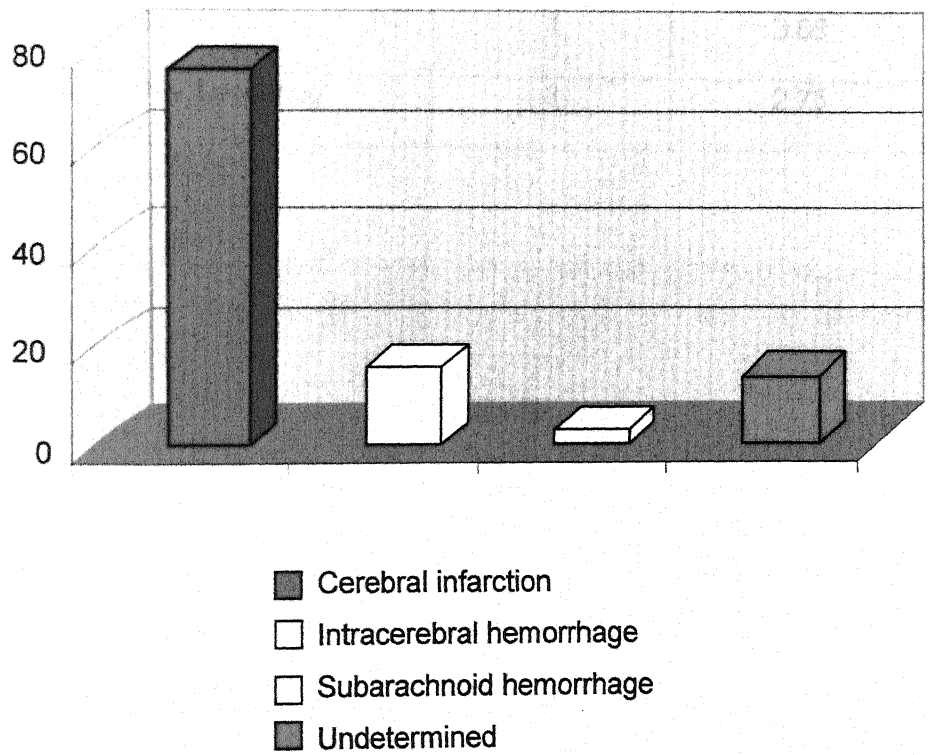


Table III

Distribution of clinical features

Clinical features	(n=109)	Percentage
Hemiparesis/hemiplegia	84	77.06
7th nerve palsy	42	38.5
Speech disorder	35	32.11
Comatosed	19	17.43
Convulsions	4	3.66
Headache &vomiting	3	2.75

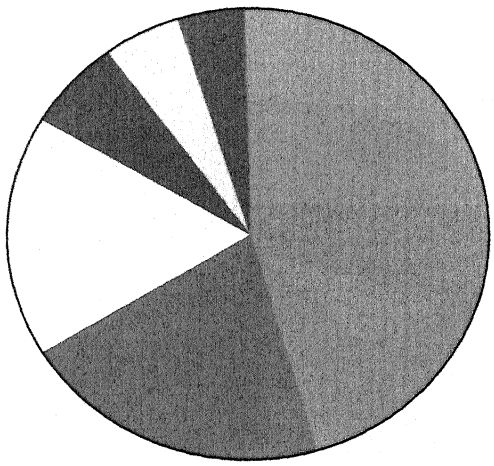
Table IV

Distribution of stroke subtypes

Stroke subtype	(n)	Percentage
Cerebral infarction	76	69.72
Intracerebral hemorrhage	16	14.67
Subarachnoid hemorrhage	3	2.75
Undetermined	14	12.84

Table 5

Distribution of risk factors in stroke



- Hypertension
- Dyslipidemia
- History of stroke / TIA
- History of hypertension
- Diabetes Mellitus
- Valvular heart Disease
- Ischemic heart disease

Table 6

Distribution of cases according to Blood Pressure as per JNC VII

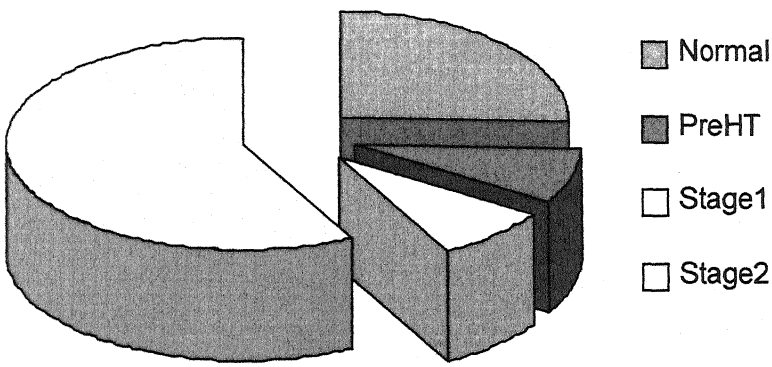


Table V

Distribution of risk factors in stroke

Risk factors	(n)	Percentage
Hypertension	71	65.13
Dyslipidemia	32	29.35
H/O stroke or TIA	14	12.84
H/O hypertension	13	11.92
Diabetes mellitus	10	9.17
Valvular heart disease	8	7.33
IHD	7	6.42

Table VI

Distribution of blood pressure of patients as per JNC VII

	(n)	Percentage
Normal	28	25.68
Prehypertensive	9	8.25
Stage1	9	8.25
Stage2	63	57.79

Table VII

Stroke outcome at 30 days

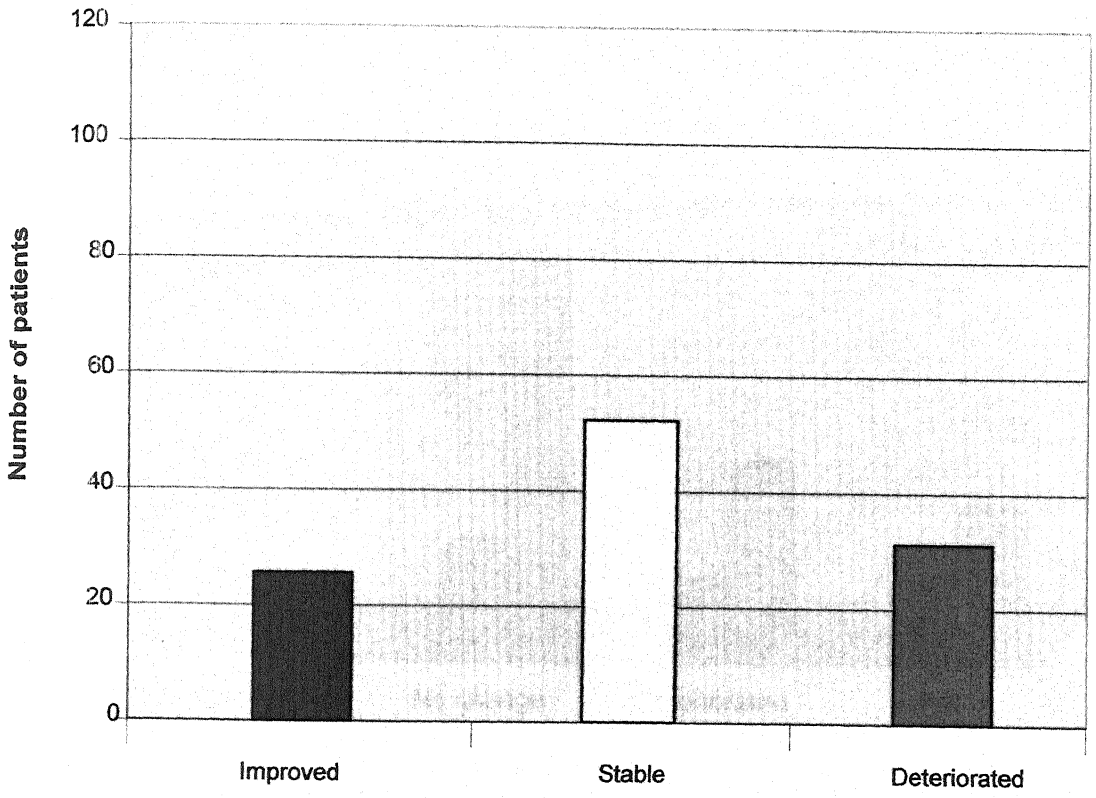


Table IX

Effect of pharmacological elevation of blood pressure on outcome in the two groups

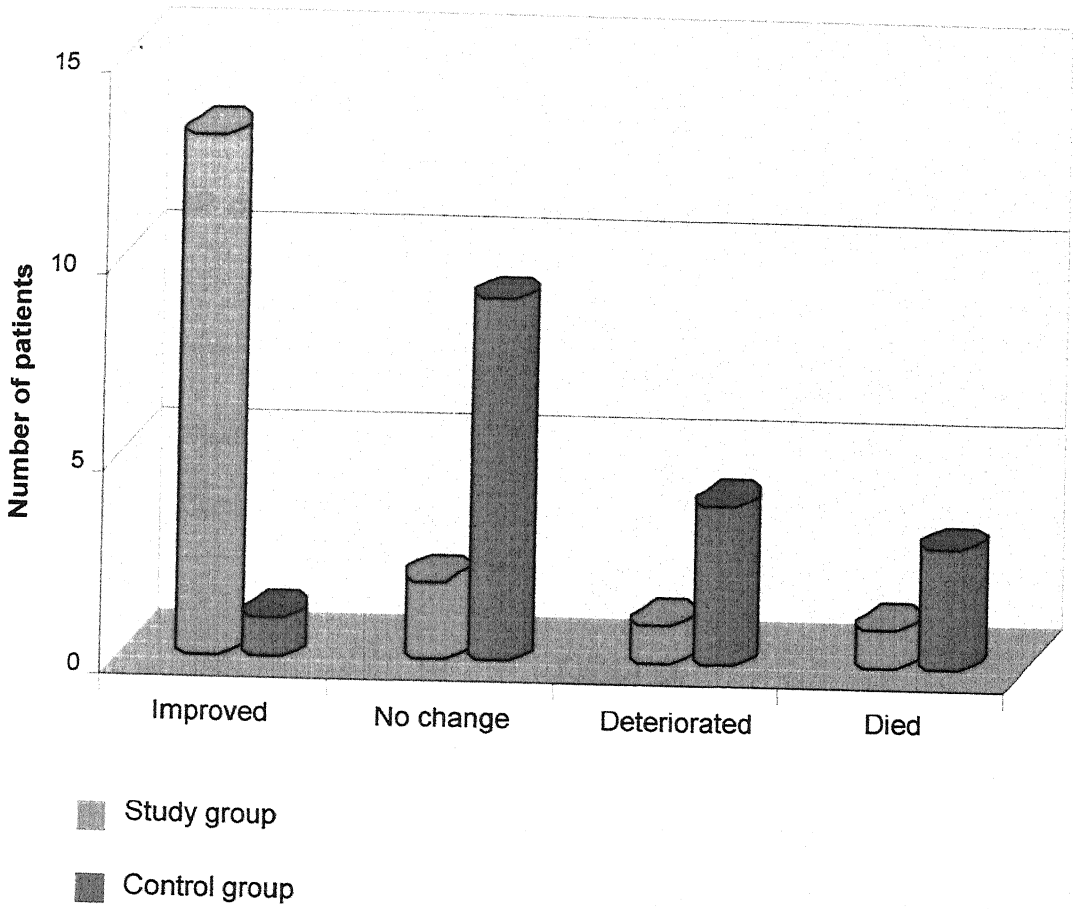


Table VII

Stroke outcome at 30 days

	'n'	%
Total Number of patients	109	
Improved	26	23.85 %
Stable	52	47.7%
Deteriorated	31	28.44%

Table VIII

MABP of study group (after elevation), control group after five days

	MABP	MABP after 5 days
Study group (n=17)	88.63 ± 13.49	98.98 ± 7.6
Control group (n=17)	90.78 ± 12.2	89.37 ± 9.77

Table IX

Effect of pharmacological elevation of blood pressure on outcome in the two groups.

	Improved	No change	Deteriorated	Death
Study group (n=17)	13	2	1	1
Control (n=17)	1	9	4	3

Table XI

Outcome of Blood Pressure lowering in the study group

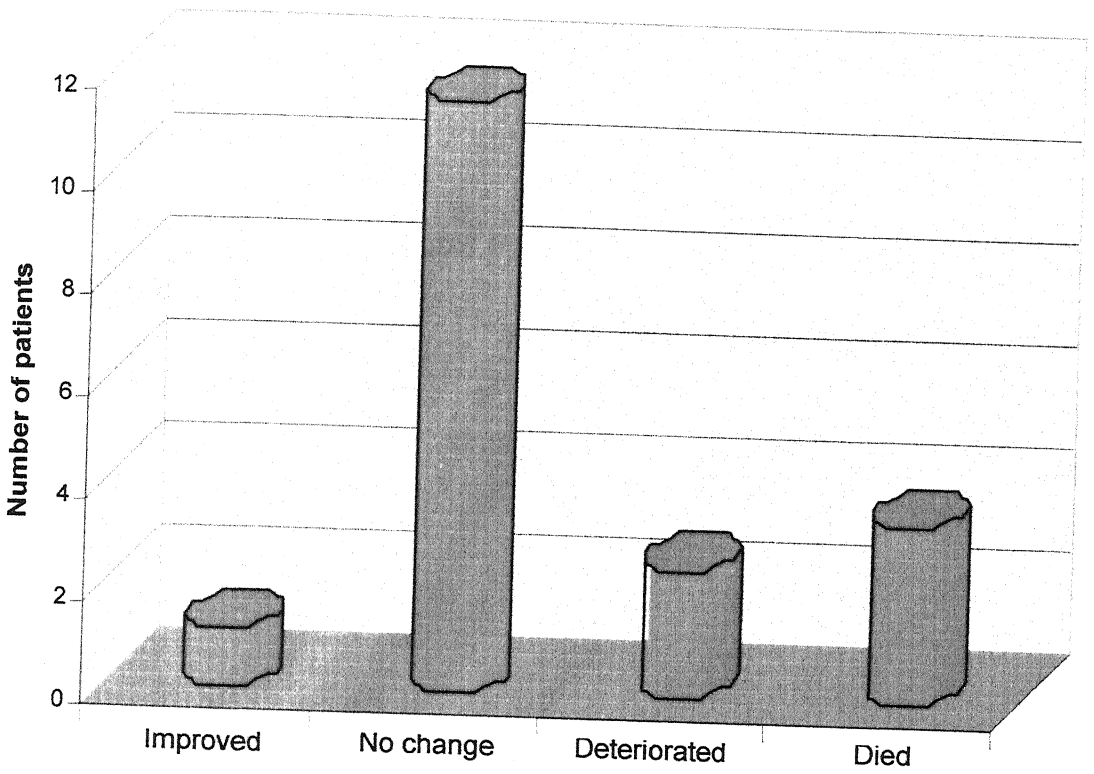


Table X

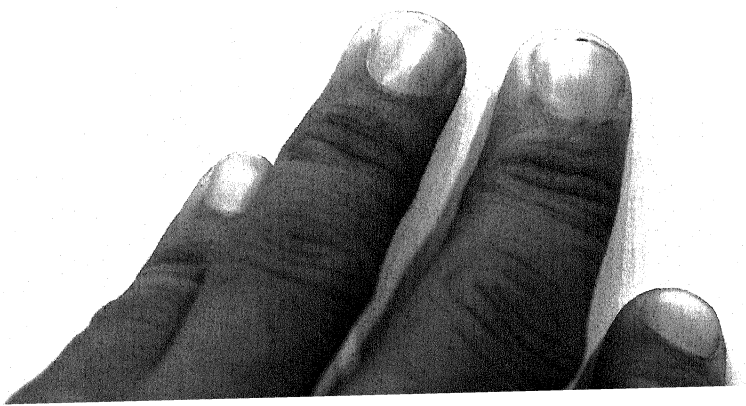
MABP of the study group (group 2) before and after lowering of blood pressure

	MABP	MABP after lowering
Study group (n=17)	160.27 ± 7.46	129.57 ± 4.58

Table XI

Outcome of Blood Pressure lowering in the study group.

	Improved	No change	Deteriorated	Died
Study group (n=17)	1	11	2	3



Discussion

Discussion

The study was conducted in the department of medicine of M.L.B. Medical College, Jhansi from the period August 2003 to September 2004. The cases were patients coming to medicine OPD and in the emergency being admitted in the medicine ward as cases of stroke.

One hundred and nine cases of stroke were included in the study. These were aged between 20 to 95 years, average age 60.95 ± 14.32 years; the maximum number of cases were in 7^{th} and 8^{th} decade thus reflecting an increase in the incidence of stroke with age which was similar to that found by Maria AK et al.[79] The male preponderance and higher association with hypertension (65%) was consistent with other studies.[80-82] 77(70.6%) were males and 32 (29.35%) females, M:F 2.4:1which increased to 3.78:1 in stroke in elderly (>60 yrs). The ratio of stroke in young (<40 yrs) was M:F 1:3.

The mean systolic blood pressure on admission was 158±42.3 mmHg and the mean diastolic blood pressure was 90.5±19.8 mmHg. The higher association of hypertension with PICH (100%) than in CI (47.36%) was comparable with a study conducted in AIIMS[83] and a study in Himachal Pradesh entitled Stroke in High Altitude by Prashar

BS et al. past H/O stroke/TIA (12.8%) was comparable with Kaur et al.[84] Incidence of diabetes mellitus 10(9.17%) in my study was similar to three studies including the one in Kashmir valley.[79,81,82] Incidence of rheumatic heart disease 8 (7.3%) was also similar to other studies done in the northern region.[80,84,85] Incidence of coronary artery disease 7(6%) was higher than stroke at high altitude

On CT scan 76(69.7%) were infarcts, 16(14%) intracerebral hemorrhage, 3 (2.75%) subarachnoid hemorrhage and 14 (13%) were undetermined. Lesions of right side of brain were more common (51R, 33L). Parietal lobe (50%) was most commonly involved site, extensive lesions (>2 areas involved) in 41% was next most common. Cerebellum (3%), temporal (3%) and frontal (2%) was other area involved.

Eighty-four (77%) had hemiplegia/hemiparesis, 42 (38%) 7th nerve palsy, 35 (32%) speech disorder, 19 (17%) were unconscious, 4 (3.66%) had convulsions and 3 (2.75%) had headache and vomiting. Hemiparesis, 7th nerve palsy and speech disorders were equally distributed between CI and PICH, whereas headache, vomiting and altered sensorium more common in PICH.

Dyslipidemia was seen in 32 (29.35%), and previous H/O hypertension in 13 (11.9%).

The admission blood pressures as per JNC VII were as follows: 28 (25.68%) normal, 9 (8.25%) prehypertensive, 9 (8.25%) stage1, and 63 (57.59%) stage 2.

During 30 days following stroke the outcome was that 26 of the patients improved with only mild residual disability, while 48 of them showed no signs of improvement and there disability was stationary. Fifty-two of them deteriorated and worsening course during their stay in hospital and there after. 10 of the patients died of which 8 during the first 24 –72 hours of their admission.

The second part of my study involved pharmacological elevation of blood pressure in patients who developed a fall in MABP within 48 hrs of admission along with worsening neurological deficit as adjudged by Mathew score.

Thirty-four patients were selected who showed a fall in their blood pressure when compared to that at admission. Seventeen (every alternate starting from the first) comprised of study group1 who underwent elevation of blood pressure. MABP of this group was 88.63$\pm$13.49 that increased to 98.98$\pm$7.6 after elevation of blood pressure for five days. In the control group the MABP after 48 hours was 90.78$\pm$12.2 and after 5 days it was 89.37$\pm$9.77 ($p<.01$). In the study group after 5 days 13 improved while in the control group this figure was only one, 2 of them showed no change one of them

deteriorated while 1 one of them died. The control group showed results quite opposite to study group, in this improvement was seen in only 1 of the patients, 9 showed no change, 4 deteriorated while 3 died during the study. This was consistent with similar study undertaken by Guy Rordorf, Steven C, Cramer in their study of pharmacological elevation of blood pressure in acute stroke.[77]

This suggests that, elevation of blood pressure can be performed without undue morbidity and mortality in acute stroke patients. Furthermore, in selected patients raising the BP was consistently associated with rapid improvement in neurological deficits.

These associations suggest that patients requiring pressor agents to preserve neurological function might have more extensive brain regions with maximally dilated vascular beds in which local CBF is sensitive to BP.

The length of time during which group I patients depended on hypertensive therapy to maintain there level of neurological function varied considerably. Natural thrombolysis or improved collateral flow may have occurred to eventually obviate the need for increased BP.

Parallel clinical observations have been observed in patients with vasospasm after SAH. In vasospasm after SAH, induced hypertension is thought to improve leptomeningeal collateral flow and

improve CBF in the maximally dilated vascular bed.[45] Patients with focal brain ischemia treated with vasopressor drugs have been described.[40,42,86] The largest cohort of patients treated with hypertensive therapy was reported by Wise et al[42] in 1972. Thirteen patients were treated with pressors, 5-showed improved neurological status immediately after increased BP, in 3 of the 5, significant recovery was maintained after the immediate postischemic period. Other series reported improved neurological function after BP was raised in association with volume expansion after LMW dextran.[87,88]

Indeed phenylephrine-induced hypertension has been reported to improve oxygen metabolism on PET in a stroke patient[89] and CBF in patients with vasospasm after SAH.[45] Hypertensive therapy may also act by preventing postischemic hypoperfusion.[90-97]

The paucity of differences seen in baseline and in morbidity/mortality between groups using the drug and those not suggest that phenylephrine use is safe in the setting of acute stroke. However, deleterious effects have been observed in some instances,[98-100] including increased blood-brain barrier permeability[101] and vasogenic edema.[99] In my study there was no increased risk for development of neurological complications due to the use of systemic hypertensive therapy.

Phenylephrine was selected as vasopressor agent of choice in our patients because (1) cerebral vessels have low density of alfa1 receptors so that phenylephrine does not produce significant direct cerebral vasoconstriction, and (2) as a pure alfa 1 agonist does not cause tachycardia or tachyarrhythmias. However, it can cause direct vasoconstriction in the coronary artery circulation and increased afterload and may contribute to congestive heart failure, cardiac ischemia, renal insufficiency, and gastrointestinal ischemia.[102]

Seventeen patients were included in the group 2 which shoed an elevation or had blood pressure >220/110 mmHg. All 17 patients underwent lowering of blood pressure. Lowering of blood pressure in acute ischemic stroke ($p<.001$) although very significant produced changes that could not be labeled as significant in any direction. Following lowering of blood pressure by nimodipine and other antihypertensive drugs produced no change in outcome of 11 of the patients, 1 of them improved, 2 deteriorated, and 3 died during the five days.

Conclusion

Conclusion

The following conclusions can be drawn from the present study:

- The average age of the population experiencing stroke was 60.95+14.32 years with the maximum cases were in the 7th and the 8th decade.
- Males clearly out numbered the females with the M:F ratio of 2.4:1 this increased to 3.78:1 in >60yrs but the ratio reversed in <30yrs, 1:3.
- Hemiparesis/hemiplegia more of the right side was present in maximum number of cases with 7th nerve palsy the next most common clinical feature followed by speech disorders, a great number of cases were unconscious and a few had convulsions, headache and vomiting.
- Hypertension was the most common risk factor present in majority of the cases, dyslipidemia the next most common, previous history of stroke/TIA, H/O hypertension, diabetes mellitus, Valvular heart disease, and evidence of ischemic heart disease in decreasing order of frequency.

- Cerebral infarction was the most common lesion on CT scan followed by hemorrhage and very few had Subarachnoid hemorrhage.
- Majority of cases admission blood pressure fell under stage 2 hypertension as per JNC VII.
- Stroke outcome at 30 days showed that 26 of the cases improved while majority showed no change and the rest deteriorated.
- Blood pressure elevation of the subjects resulted in better outcome in majority.
- Lowering of blood pressure had no change in outcome of majority of the cases.

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MASTER CHART

MRD No	name	Age	Sex	symptoms	BP at Adm	BP at 48 hrs	CT Scan	HT	DM	Dyslipid	Prev Stroke	H/O HT	IHD	RHD
11988	Mannulal	70	M	unc	180 / 100	186 / 100	Hemmorhg	Yes	No	No	Yes	No	No	No
12110	Narada	60	F	LHg,7th n.	160 / 90	130 / 80	Infarct	Yes	Yes	Yes	No	No	No	No
12148	Chandan Sg	40	M	LHg, Sp dis	180 / 80	186 / 84	Infarct	Yes	No	Yes	No	No	No	No
12197	Guldad	70	M	Unc	220 / 120	224 / 128	Hemmorhg	Yes	Yes	Yes	No	Yes	No	No
12343	Prem sg	73	M	LHp, 7th n	110 / 70	100 / 60	Infarct	No	No	No	No	No	No	No
12871	B.S. Verma	63	M	Unc	230 / 130	240 / 136	Hemmorhg	Yes	No	No	No	No	No	No
13123	Harbi	80	F	LHp	180 / 80	186 / 84	Uknown	Yes	No	Yes	No	No	No	No
13170	A.P.Shar	62	M	LHp, 7th n	176 / 90	180 / 100	Infarct	Yes	No	No	No	No	No	No
13318	batan lal	48	M	LHp, 7th n	160 / 90	130 / 90	Infarct	Yes	No	No	No	No	No	No
13382	daya ram	45	M	RHg, Sp dis, 7th n	156 / 90	118 / 80	Uknown	Yes	No	Yes	No	No	No	No
13805	pushpa bai	40	F	LHp,unc	70 / 50	100 / 50	Infarct	No	No	Yes	Yes	Yes	No	No
14123	Ramale	80	M	RHg,Sp dis, 7th n	150 / 110	160 / 108	Infarct	Yes	No	No	No	No	No	No
14568	Dheeraj	60	M	RHp, Sp dis	160 / 80	168 / 90	Infarct	Yes	No	No	No	No	No	No
14623	Mathura	65	M	RHp, Sp dis, 7th n	110 / 80	100 / 70	Infarct	No	No	Yes	No	No	No	No
14621	karan	30	M	RHg, Sp dis, 7th n	100 / 60	90 / 60	Infarct	No	No	No	No	No	No	Yes
14760	gopi	77	M	LHp,7th n	140 / 90	150 / 90	Infarct	No	Yes	Yes	Yes	No	Yes	No
15178	brij rami	55	M	unc, conv	230 / 126	240 / 128	Hemmorhg	Yes	No	No	Yes	Yes	No	No
15696	kayshiya	85	F	RHg, Sp dis, 7th n	180 / 98	170 / 90	Hemmorhg	Yes	No	No	No	No	No	No
16194	chiroja bai	75	F	LHp	100 / 60	120 / 70	Infarct	No	No	Yes	No	No	No	No
16236	lachi	68	F	RHp, Sp dis, 7th n	158 / 70	168 / 80	Infarct	Yes	No	Yes	No	No	No	No
16485	bhaya lal	50	M	LHp	158 / 100	120 / 70	Infarct	Yes	No	No	No	No	No	No
16626	marium	48	F	unc, conv	228 / 120	220 / 110	Hemmorhg	Yes	No	No	No	No	No	No
16676	manwati	55	M	RHp, 7th n	100 / 80	120 / 80	Infarct	No	No	No	No	No	No	No
16487	ramkali	72	F	RHg, 7th n	240 / 130	230 / 120	Infarct	Yes	No	Yes	No	No	No	No
17041	ram sewak	60	M	unc	200 / 100	220 / 120	Hemmorhg	Yes	No	Yes	No	No	No	No
17171	uttam chandra	76	m	unc	198 / 100	180 / 100	Infarct	Yes	No	No	No	No	Yes	No
17332	narwada	40	F	RHp, Sp dis, 7th n	110 / 70	100 / 70	Infarct	No	No	Yes	Yes	No	No	Yes
17466	ramwati	60	F	RHp, Sp dis, 7th n	132 / 80	140 / 90	Infarct	No	No	No	No	No	No	No
17494	jalam	60	M	headache,vomiting	110 / 80	120 / 70	Infarct	No	No	No	No	No	No	No
17621	khajju	80	M	RHp, 7th n	176 / 110	156 / 110	Uknown	Yes	No	No	No	No	No	No
18215	subbir khan	65	M	headache,vomiting	138 / 90	130 / 80	SAH	No	No	Yes	No	No	No	No
18607	ram nath	70	M	LHp	238 / 140	230 / 140	Infarct	Yes	No	No	No	No	No	No
18468	amir baksh	60	M	LHp	150 / 90	130 / 90	Uknown	Yes	No	Yes	No	No	No	No
18728	sita ram	63	M	RHg, 7th n	110 / 70	100 / 60	Infarct	No	No	No	No	No	No	No
18793	nathu ram	65	M	RHg, Sp dis	160 / 80	170 / 80	Uknown	Yes	Yes	Yes	Yes	Yes	No	No
18298	ram kishore	65	M	RHp, 7th n	110 / 80	120 / 90	Infarct	No	No	Yes	No	No	No	No
19111	param lal	50	M	RHp, Sp dis	150 / 100	160 / 100	Uknown	Yes	No	No	No	No	No	No
19366	lachhi ram	65	M	LHp	200 / 100	220 / 130	Hemmorhg	Yes	No	No	No	No	No	No

19427	ram das	60	M	RHg, 7th n	160 / 94	168 / 100	Infarct	Yes	No	Yes	No	No	No	No
19516	ghamandi	65	M	unc	200 / 114	210 / 110	Hemmorhg	Yes	No	No	No	No	No	No
19970	sumu	85	M	RHg, conv	226 / 120	230 / 120	Hemmorhg	Yes	No	No	No	No	No	No
20056	kashi	70	M	RHp, 7th n	118 / 86	100 / 70	Infarct	No	No	No	No	No	No	No
20267	premwati	70	F	broca's aphasia	148 / 88	120 / 70	Infarct	Yes	Yes	Yes	Yes	No	No	No
20585	lalita bai	20	F	RHg, Sp dis	238 / 130	240 / 120	Infarct	Yes	No	No	No	No	No	No
20880	asha rani	75	F	LHp, 7th n	170 / 80	120 / 80	Infarct	Yes	No	No	No	No	No	No
21380	bari bai	35	F	RHp	120 / 70	110 / 70	Infarct	No	No	No	No	No	No	Yes
48	maarti	20	F	RHp, Sp dis	100 / 60	118 / 60	Infarct	No	No	No	No	No	No	Yes
295	bhuwan bai	55	F	RHp, Sp dis, 7th n	160 / 90	130 / 80	Infarct	Yes	No	No	No	No	No	No
299	Gaffar	55	M	unc	248 / 130	248 / 136	Infarct	Yes	No	No	Yes	Yes	No	No
419	ramcharan	54	M	LHg, 7th n	190 / 100	200 / 100	Hemmorhg	Yes	No	No	No	No	No	No
548	kastoori	40	F	LHp, 7th n	190 / 70	196 / 70	Infarct	Yes	No	Yes	No	No	No	Yes
557	maharaj sg	50	M	unc	220 / 100	226 / 110	Hemmorhg	Yes	No	No	No	No	No	No
669	narayan das	70	M	LHp	160 / 110	130 / 88	Infarct	Yes	No	No	No	No	No	No
766	sunaina	56	F	RHp, 7th n	154 / 90	120 / 90	Uknown	Yes	No	No	No	No	No	No
975	bihari lal	65	M	unc	160 / 90	130 / 80	SAH	Yes	No	No	No	Yes	No	No
1055	bhagwati	65	M	broca's aphasia	150 / 80	120 / 70	Uknown	Yes	No	Yes	No	No	No	No
1263	manto bai	60	F	Lhp	160 / 90	120 / 80	Infarct	Yes	No	No	No	No	Yes	No
1742	kalicharan	62	M	RHg, Sp dis	210 / 120	220 / 130	Uknown	Yes	No	No	No	No	No	No
1924	b khattori	50	F	unc	142 / 90	130 / 90	Infarct	Yes	No	No	No	Yes	No	No
1925	nathu	45	M	headache,vomiting	160 / 80	120 / 80	Infarct	Yes	No	Yes	Yes	Yes	No	No
2073	kalka	65	M	RHp, Sp dis, 7th n	120 / 70	120 / 70	Infarct	No	No	No	No	No	No	No
2404	lambey	50	M	RHp, 7th n	160 / 88	138 / 80	Infarct	Yes	No	Yes	No	No	No	No
2661	abhai	55	M	RHp, 7th n	100 / 60	120 / 70	Infarct	No	No	No	No	No	Yes	No
2681	vinod kr	40	M	LHp	180 / 100	190 / 100	Infarct	Yes	No	No	No	No	No	No
3179	sumita devi	65	F	RHp, Sp dis, 7th n	118 / 90	130 / 90	Infarct	No	No	No	No	No	No	No
3275	ram charan	65	M	LHp	170 / 90	128 / 90	Uknown	Yes	No	No	No	No	No	No
3748	parsi	70	M	RHp, Sp dis	124 / 80	130 / 80	Infarct	No	No	Yes	No	No	No	No
4706	anita	32	F	RHp, 7th n	108 / 60	120 / 60	Infarct	No	No	No	No	No	No	Yes
5510	govind sg	60	M	RHp, 7th n	180 / 80	170 / 90	Infarct	Yes	No	No	No	No	No	No
5591	charmu	75	M	LHp	170 / 90	180 / 80	Infarct	Yes	No	No	No	No	No	No
6027	ram pyari	65	F	RHp,aphasia, 7th n	170 / 110	130 / 90	Infarct	Yes	No	No	No	No	No	No
6074	kapoori	64	F	LHp	170 / 90	150 / 90	Infarct	Yes	No	No	No	No	No	No
6121	shanti	75	F	LHg	180 / 110	190 / 110	Infarct	Yes	Yes	Yes	Yes	Yes	No	No
6327	nababuddin	65	M	RHg, Sp dis	200 / 108	210 / 110	Hemmorhg	Yes	No	No	No	Yes	No	No
6844	mulu	45	M	RHp, aphasia	128 / 70	140 / 70	Infarct	No	No	No	No	No	Yes	No
6868	jai jai ram	45	M	broca's aphasia	152 / 100	138 / 90	Infarct	Yes	No	No	No	No	No	No
7052	kalawati	70	F	RHp, Sp dis	110 / 80	120 / 80	Infarct	No	No	No	No	No	No	No
7917	munnalal	45	M	LHg	180 / 110	170 / 110	Uknown	Yes	Yes	No	No	No	No	No

7909	prem narayan	65	M	RHg, Sp dis	98 / 60	110 / 70	Infarct	No	No	No	No	No	No	No
8337	janath yadav	60	M	RHg, 7th n	164 / 100	128 / 100	Infarct	Yes	No	No	No	No	No	No
8348	jugal kishore	85	M	RHg, Sp dis	150 / 100	130 / 80	Infarct	Yes	No	No	No	No	No	No
8584	raja bai	50	M	RHp, 7th n	130 / 76	138 / 80	Infarct	No	No	No	No	No	No	No
8793	bari bau	85	F	RHg, Sp dis	190 / 100	200 / 100	Infarct	Yes	No	No	No	No	No	No
8967	tijjan	80	M	RHp, 7th n	180 / 100	190 / 100	Infarct	Yes	Yes	Yes	Yes	No	No	No
9184	laloo rampal	60	M	unc	200 / 110	230 / 130	SAH	Yes	No	No	No	No	Yes	No
9788	ram lal	64	M	RHp, 7th n	110 / 60	130 / 70	Infarct	No	No	No	No	No	No	No
9846	nahu ram	65	M	unc	180 / 100	198 / 100	Hemmorhg	Yes	No	Yes	No	No	No	No
10287	ghyan sg	50	M	RHp, Sp dis	100 / 70	120 / 70	Infarct	No	No	No	No	No	No	No
10713	tulsi das	73	M	unc	210 / 120	220 / 130	Hemmorhg	Yes	No	Yes	No	No	No	No
10837	lachchho	55	F	unc	226 / 100	240 / 110	Hemmorhg	Yes	No	No	No	No	No	No
10905	mahesh	45	M	RHg, Sp dis	120 / 60	130 / 70	Infarct	No	No	No	No	No	No	No
11242	khuman	50	M	LHp, 7th n	100 / 56	110 / 60	Infarct	No	No	No	No	No	No	No
11991	maya	40	F	RHg, 7th n, conv	126 / 70	130 / 80	Infarct	No	No	No	No	No	No	Yes
12403	hazari lal	70	M	unc	190 / 100	130 / 80	Uknown	Yes	No	No	No	No	No	No
13229	sukh lal	75	M	RHg	100 / 80	120 / 70	Infarct	No	No	No	No	No	No	No
13500	arvind	48	M	LHg, 7th n	120 / 80	130 / 80	Infarct	No	No	No	No	No	No	No
13520	mukundi	76	M	unc	180 / 116	190 / 120	Uknown	Yes	No	No	No	No	No	No
13602	bhaggu	70	M	RHg, Sp dis	110 / 70	120 / 70	Infarct	No	No	No	No	No	No	No
13998	raj kumar	95	M	LHp, Sp dis	160 / 100	120 / 80	Infarct	Yes	No	Yes	Yes	Yes	No	No
14327	raj kumar	75	M	LHg	100 / 80	90 / 60	Uknown	No	No	No	No	No	No	No
14869	ram swarop	60	M	RHg, Sp dis	170 / 90	180 / 100	Infarct	Yes	No	No	No	No	No	No
14989	marayan giri	70	M	unc	178 / 90	190 / 90	Infarct	Yes	No	No	No	No	No	No
15012	prabhu dayal	70	M	LHp,7th n	160 / 100	124 / 80	Infarct	Yes	No	No	Yes	Yes	Yes	No
16035	janki prasad	70	M	RHg, 7th n	116 / 70	130 / 80	Infarct	No	No	No	No	No	No	No
16370	hari sg	25	M	LHg	90 / 60	80 / 60	Infarct	No	No	No	No	No	No	Yes
16391	maryan sg	78	m	RHp, 7th n	122 / 84	100 / 80	Infarct	No	No	Yes	No	No	No	No
16432	udai bha	70	F	RHp, Sp dis	280 / 150	240 / 130	Infarct	Yes	Yes	Yes	No	No	No	No
16449	kamala devi	65	F	LHp	110 / 80	90 / 60	Infarct	No	No	No	No	No	No	No
15640	dharam bai	72	F	LHg	190 / 80	220 / 120	Infarct	Yes	No	Yes	Yes	Yes	No	No

BP : Blood Pressure, M : Male, F : Female, LHg : Left hemiplegia, LHp : Left hemiparesis, RHg : Right Hemiplegia, RHp : Right hemiparesis, unc : unconsciousness, Sp dis : Speech disorder, Conv : Convulsions, 7th n : 7th nerve palsy, Adm : Admission, HT : Hypertension, DM : Diabetes Mellitus, IHD: Ischemic Heart Disease, RHD : Rheumatic Heart Disease, Dyslipid : Dyslipidemia, MABP : Mean Arterial Blood Pressure

Group 1

Study Group				Control Group			
	BP at 48 hours		After elevation of BP (5 days)		BP at 48 hours		After 5 days
No		MABP		MABP	Sl.No		MABP		MABP
1	100 / 60	73.33	148 / 80	102.67	1	130 / 80	96.67	110 / 70	83.33
2	118 / 80	92.67	150 / 90	110.00	2	130 / 90	103.33	130 / 80	96.67
3	100 / 70	80.00	128 / 60	82.67	3	100 / 50	66.67	120 / 60	80.00
4	120 / 70	86.67	124 / 80	94.67	4	90 / 60	70.00	110 / 70	83.33
5	130 / 90	103.33	120 / 80	93.33	5	100 / 70	80.00	110 / 80	90.00
6	100 / 70	80.00	126 / 70	88.67	6	100 / 60	73.33	108 / 80	89.33
7	110 / 70	83.33	138 / 80	99.33	7	120 / 70	86.67	118 / 70	86.00
8	130 / 88	102.00	140 / 80	100.00	8	130 / 80	96.67	136 / 90	105.33
9	130 / 80	96.67	128 / 90	102.67	9	120 / 90	100.00	128 / 70	89.33
10	120 / 80	93.33	130 / 70	90.00	10	120 / 70	86.67	128 / 80	96.00
11	138 / 80	99.33	140 / 90	106.67	11	130 / 90	103.33	136 / 80	98.67
12	130 / 90	103.33	138 / 80	99.33	12	128 / 90	102.67	120 / 90	100.00
13	128 / 100	109.33	140 / 90	106.67	13	138 / 90	106.00	120 / 90	100.00
14	130 / 80	96.67	146 / 90	108.67	14	130 / 80	96.67	128 / 80	96.00
15	90 / 60	70.00	128 / 80	96.00	15	120 / 80	93.33	100 / 60	73.33
16	80 / 60	66.67	136 / 90	105.33	16	124 / 80	94.67	96 / 60	72.00
17	90 / 60	70.00	128 / 80	96.00	17	100 / 80	86.67	100 / 70	80.00
	Total	1506.67	Total	1682.67		Total	1543.33	Total	1519.33
	Mean	88.63	Mean	98.98		Mean	90.78	Mean	89.37
	S.D	13.49	S.D	7.60		S.D	12.20	S.D	9.77

Group 2

Study group after 48 hrs			BP after lowering (5 days)		
	MABP (X1)			MABP (X2)	X1 - X2
224 / 128	160.00		190 / 100	130.00	30.00
240 / 136	170.67		176 / 90	118.67	52.00
240 / 128	165.33		176 / 98	124.00	41.33
220 / 110	146.67		198 / 100	132.67	14.00
230 / 120	156.67		190 / 104	132.67	24.00
220 / 120	153.33		180 / 100	126.67	26.67
230 / 140	170.00		200 / 98	132.00	38.00
220 / 130	160.00		190 / 100	130.00	30.00
230 / 120	156.67		186 / 110	135.33	21.33
240 / 120	160.00		200 / 100	133.33	26.67
248 / 136	173.33		196 / 96	129.33	44.00
226 / 110	148.67		190 / 100	130.00	18.67
220 / 130	160.00		200 / 104	136.00	24.00
230 / 130	163.33		180 / 104	129.33	34.00
220 / 130	160.00		198 / 100	132.67	27.33
240 / 110	153.33		190 / 96	127.33	26.00
240 / 130	166.67		180 / 94	122.67	44.00
Total	2724.67		Total	2202.67	522.00
Mean	160.27		Mean	129.57	30.71
S.D	7.46		S.D	4.58	10.16